



***Vibrio cholerae* Incursion in Africa, the Journey So Far**

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Authors' contributions

This work was carried out in collaboration among all authors. Author TJE designed the study, performed the statistical analysis, wrote the protocol and wrote the first draft of the manuscript. Authors CIM and BEEA managed the analyses of the study. Authors TJE and ICU managed the literature searches. All authors read and approved the final manuscript.

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ABSTRACT

Indeed, a host of the plenty of reports about pathogenic *Vibrios*, have been from African researchers. And they assert that this severe diarrhea causing agent originated from Asia thousands of years ago and spread (first, via the sea route) affecting particularly the coastal towns and fishing villages, before moving to other parts of the world. Following the primary cholera outbreak of 1868, *Vibrio cholera*, appeared in the Atlantic coast of West Africa. The pathogen then invaded African countries chronologically beginning with Guinea, then Sierra Leone, Liberia, Cote d'ivoire, Mali, Togo, Dahomey, Upper Volt (Burki Na-faso) and finally Nigeria (Lagos) and Niger in December, 1970. Various serogroups (O139 and O1 with biotypes Classical and El Tor) and Serotypes of O1 (Ogawa, Inaba and Hikojima) and recently, the O395 strain have been reported; especially from outbreaks reported from hotspots that are close to riverine areas. This suggests that these emergent pathogenic species originate from around water environments probably from the non-pathogenic strains. This condition is likely harnessed by Lateral Gene Transfer (LGT), which is seen to occur usually between pandemic *V. cholerae* and environmental strains; a

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situation that may result in the creation of new pandemic strains. Therefore, in order to better understand and appreciate the evolution of the *Vibrio cholerae* strains that are involved in epidemics, and the relationship between the species causing particular epidemics in different regions of Africa, a study of the molecular picture of the environmental strains and the mechanisms by which the pathogenic *Vibrio cholera* strains appear and diffuse from these strains is necessary. This review seeks to trace the origin and spread route of *Vibrio cholerae* strains causing epidemics in different regions of the African continent (Nigeria in particular) with the aim of establishing relationships between the strains causing epidemics in these regions. This will help in the development of better intervention strategies to contain the disease.

Keywords: *Vibrio; cholera; incursion; Africa; journey.*

1. INTRODUCTION

In Africa, especially in countries like Nigeria, uncontrolled location of the majority of people in areas with limited basic necessities is harnessed by Rural to Urban migration and/or population growth [1]. In such areas, potable water, good toilets and waste disposal systems etc, consequently, are looked at as commodities set aside basically for the wealthy people instead of being the basic necessities which they are. Thus a host of this population use streams, springs and wells [2] which normally are supplied by surface runoff, containing untreated wastes from homes, workshops, factories and industries [1]. Thus, microbiologically, the quality of these polluted sources of water is usually highly compromised [1].

Therefore countries (Nigeria for example) which are situated along the water banks like the Atlantic coast line of west and central Africa have been shown, using epidemiologic history to be endemic for *Vibrio species* [3]; especially the deadly diarrheal disease (cholera)causing *Vibrio Cholerae* [4].

Vibrio cholerae spread from Asia where it was first discovered, to other parts of the world [3,5], affecting particularly the coastal towns and fishing villages [6]. It gained foothold within these regions where it arrived with the tribal behaviors of the population – funeral ceremonies, movement of people within and between countries, business [7,6], outdoor relaxation, fishing navigation, source of drinking water and food [8,9,10].

The arrival of *Vibrio cholerae* to the Atlantic coast of West Africa in 1868 ushered in the first cholera outbreak to the region. Since then, *Vibrio* have been reported in South-East and South-South Nigeria [11,2,12,13,14,15,16], South west Cameroon [17], as well as in other African countries [18].

From the cholera outbreaks that have been reported from localities close to riverine environments, different serotypes have been isolated [19]. This suggests that these water environments may be good reservoirs that generate new strains of infectious *Vibrio cholerae* from the non-infectious strains located in these areas. This development may most likely be by Lateral Gene Transfer (LGT) [20].

The molecular picture of the environmental strains of *Vibrio cholerae* in the African regions, when compared with the strains reported from epidemics, may give a clue to the spread route of the *Vibrio cholerae* causing these epidemics in Africa.

The Physiology of *Vibrio* species *Vibrios* are bacterial species that have been classified in the genus of comma shaped bacilli measuring 0.5–0.8 ×1.4–2.6 µm in diameter [21]. These members of the gamma group of Proteobacteria are classified in the family *Vibionaceae* [21]. When grown in a liquid medium, they do not produce spores, but produce motile, straight or curved, single, rigid, and Gram negative rods, with a monotrichous continuous sheathed flagellum [22].

Not less than 34 recognized species of *Vibrio*, both non-pathogenic aquatic strains and epidemic cholera (*V. cholerae*, *V. parahaemolyticus* and *V. vulnificus*) have been identified [8].

During in-vitro culture on solid media, *Vibrios* of the species *alginolyticus*, *campbellii*, *harveyi*, *parahaemolyticus*, and some strains of *fluvialis* develop, unsheathed amphitrichous flagella [22]. *Vibrio in the species* *alginolyticus*, *parahaemolyticus* and *proteolyticus* develop long cells with swarming capabilities on solid enriched media [23]. Their metabolic activities are more like those of the *Enterobacteriaceae* family [19, 24,25,26].

It has been observed that for all the *V. cholerae* species to grow luxuriantly, some amount of NaCl must be added to the medium and that NaCl concentrations of at least 600–700 mM must be added to the medium before *V. costicola* colonies can grow. More so, it was also noted that *Aliivibrio logei* comb. Nov (formally *V. logei*) and *V. marinus* can only grow at the temperature range of 4–20°C, although majority of species will grow at the temperature range of 20°C – 40°C. However, high temperatures (60°C for 15 minutes) will kill *V. cholera* [22]. In the same work, the above researchers further reported that the pH of 10 is well tolerated by *Vibrio* species (especially *V. cholerae* and *V. metschnikovii*). It is on this basis that alkaline Peptone Water (where *Vibrios* form a thin pellicle), telluride supplemented media as well as Thiosulphate Citrate Bile Salt Sucrose (TCBS) Agar are used for differentiation of *Vibrio* species. They appear as 1–2 mm in diameter, circular, translucent colonies with entire margins on Nutrient Agar within 24 hours [22]. *Aliivibrio fischeri* gen. nov., comb. Nov (formally *V. fischeri*) and *Aliivibrio logei* comb. nov were observed to form a yellow-orange pigment after 3–4 days on complex solid media; and emitted an unusual yellow light at temperatures below 18°C but blue-green light at temperatures above 23°C. Kourany, [27] observed that *V. gazogenes* produced a pigment called prodigiosin, while *V. nigripulchritudo* produced insoluble blue-black crystals with an appearance that is characteristic of this species. They also observed that these crystals accumulate in colonies growing on basal media, even without the presence of organic growth factors. [28] The strains of El Tor biotype of *Vibrio cholera* O1 have also been observed to produce strong haemolytic reactions with the sheep red blood cells, a positive Vogues Proskauer test, agglutinate chicken erythrocytes, show resistance to group IV phages and polymyxin B [28].

Ashiru et al. [8] stated that majority of the members of this group of bacteria were positive for oxidase, Vogues Proskauer, Sucrose, D-Gluconate tests but negative for Arginine dihydrolase. Furthermore, Glucose metabolism is through the mixed acid fermentation pathway producing only acids without gas; and most strains do not ferment lactose, the exceptions being strains of *V. metschnikovii* and *V. vulnificus*. In their work in 2012, Ashiru et al. [8] observed that the sucrose fermentation confers yellow coloration to *V. cholera* colonies when

grown on TCBS agar, unlike colonies of the non-sucrose fermenting *V. mimicus* which appear green.

Strains of *V. diazotrophicus* have been reported by Singleton and Sainbury [22] to fix nitrogen while chitinolytic strains degrade chitin in aquatic ecosystems.

2. HABITAT

Sea foods are becoming very important dietary components due to the increased awareness of the health benefits associated with their nutritional values [8,29]. However, the bacteriological quality of some continues to be of great concern because, shellfish most especially, have been shown to be reservoirs for some pathogenic *Vibrio* species [9]. These *Vibrio* species naturally, inhabit estuarine, coastal waters and marine sediments throughout the world, since these areas have the appropriate salinity required for their growth [25,30]. They have been known to colonize sea foods such as corals, fish, molluscs, shrimps and zooplankton (specifically copepods) [31], as well as oyster, clams, mussels, periwinkles and prawns [9,30, 29], all of which are an embodiment of the marine food chain [32,33]. In a research carried out on these various sea foods, Ashiru et al. [8], observed shrimps were the most contaminated of all the sea foods evaluated (95.8%), this was closely followed by the crabs with 73.3% snails 44.3%, lobster 44.1%, sand crab 32.5%, fish 29.3% and craw fish 21.1% respectively.

However, [22] *V. splendidus* biotype I, *Aliivibrio fischeri* gen. nov., comb. nov and *Aliivibrio logei* comb. Nov), have been reported to colonize specialized luminous organs in certain teleost fish and squid. On the other hand, while *V. cholera* and *V. parahaemolyticus* are known to be pathogenic to man, *V. tubiashii* has been reported as a pathogen of bivalve and mollusks while *Aliivibrio logei* comb. nov also, as a pathogen of eels. In 2010, [33] reported that Copepods, are important reservoirs for pathogenic *V. cholera* O1 and O139 strains and this claim was buttressed by Rebaudet et al. [34], who showed that approximately 10^4 *V. cholerae*, organisms (about the infectious dose in susceptible individuals) may be carried by a copepods. Cultured fish and lobsters are often infected by the pathogenic strains of *V. fluvialis* and this sometimes results in huge economic losses to fish farmers [35].

Table 1. Some species of Vibrio found to be associated with common sea foods

<i>V. alginolyticus</i> Fish	<i>V. harveyi</i> Fish	<i>V. parahaemolyticus</i> Fish
<i>Vibrio</i> sp.1 Fish	<i>V. logei</i> Fish, Crab	<i>V. pelagius</i> Fish
<i>V. campbellii</i> Fish	<i>V. mediterranei</i> Fish, Prawn	<i>V. proteolyticus</i> Fish
<i>V. cincinnatiensis</i> Fish	<i>V. metschnikovii</i> Fish	<i>Vibrio</i> sp.2. Fish
<i>V. ctioteree</i> Fish	<i>V. mimicus</i> Fish	<i>V. splendidus</i> Fish
<i>V. costicote</i> Fish	<i>V. netreiqens</i> Fish	<i>Vibrio</i> sp.3 Fish
<i>V. fumissii</i> Fish	<i>V. orientalis</i> Fish	<i>V. vulnificus</i> Fish

Source: [36]

In their reports, these researchers [22] also reiterated that although *Vibrio species* are mostly encountered from sea foods, they can also be sometimes encountered in salted foods and brines.

3. ORIGIN OF CHOLERA, PANDEMICS AND SPREAD TO CONTINENTS ORDER THAN AFRICA

The first pandemic Cholera ever reported, started from Bengal in 1816. It later crossed to the rest of India by 1820 and in 1826 it spread to China and to the Caspian Sea [5]. From there, following the land and sea trade routes was transported to Russia, Western Europe, and to North America [3].

This dreaded infectious agent (*Vibrio Cholerae*) continued to spread in Europe and from 1829-1851, cholera claimed 6,536 lives in London, 20,000 in Paris and 100,000 lives in all of France. And this was as reported by Sajeew [5], the second pandemic of Cholera ever. It struck Russia, as a disaster in 1834, and Quebec, Ontario Canada and New York followed suite [37]. As the second occurrence, another major outbreak struck Europe in 1849 traced back to South-wark, through a sailor infected in 1848 [5]. This worst outbreak ever in the history of Europe left 14,137 dead in London, 5,308 in Liverpool and between 50,000 and 70,000 in England and Wales. Guynup [38] reported that a third outbreak occurred in this same part of the world in 1854 and left over 30,000 people dead in London alone while in North America, one time President of the United States, James K. Polk, became a victim. In the same report, Guynup stated that cholera spread through the Mississippi River, and killed over 4,500 in St. Louis, over 3,000 in New Orleans, thousands in New York, and hundreds of migrants on their way to Utah, Oregon, and the California.

Death from this massive flesh-eating infection (Cholera) bombarded Russia for the second time in 1852 and prolonged till 1860, claiming over a million lives [19]. They reported that this Pandemic was recorded as the third of the Cholera pandemic, and London and Chicago again lost 10,738 and 3,500 people, respectively, in 1854. Subsequently, in 1866, in an epidemic that was later traced to have originated from the East London Water Company, 5,596 people lost their lives in East Ending London. They also observed that the fourth global cholera outbreak affected mostly Europe and this was between the period of 1863 and 1875, while the fifth, occurred between 1881 and 1896 as the only major and last serious European cholera outbreaks that claimed about 8,600 lives.

However, they also observed that another pandemic occurred between 1899 and 1923 making it the sixth in the world history of cholera pandemics and this affected Russia (the most), the Balkan Peninsula and the Middle East.

According to a report by Mukhopadhyay et al. [7], another pandemic of Cholera (the Seventh), which originated in the island of Sulawesi in 1961, affected the entire Southeast Asian archipelago by the end of 1962. And by 1963 from Asia, cholera extended inward to Malaysia, Thailand, Burma, Cambodia, Vietnam, India, Bangladesh, and to Pakistan, by 1969. El Tor cholera entered the history of the outbreaks when reported in Afghanistan, Iran, Iraq, the Soviet Union and by 1970, El Tor cholera invaded the Arabian Peninsula, Syria, Jordan, Israel, South Pacific and Japan. In the same report, outbreaks were reported near Baku in 1972, Italy by 1973, Peru, South America (with an estimated 750,000 cases of cholera and 6,500 deaths), Ecuador and then Colombia in 1991.

Table 2. Cholera pandemics, 1817 till date

Pandemic	First	Second	Third	Fourth	Fifth	Sixth	Seventh	Eighth
Year	1817-1823	1829-1851	1852-1859	1863-1879	1881-1896	1899-1923	1961-Date	1992-Date
Origin		India				Sulawesi, Indonesia		Madras, India
Other Affected Areas	South-east Asia, Middle East and East Africa	South-east Asia, Middle East Europe, America and Africa				South-east Asia, Middle East Europe, South America and Africa		South-east Asia
Vibrio Strain	<i>Vibrio cholerae</i> serogroup O1 biotype classical	<i>V. cholerae</i> O1, classic				<i>V. cholerae</i> O1, El Tor		<i>V. cholerae</i> O139

Source: Adagbada et al. [2]

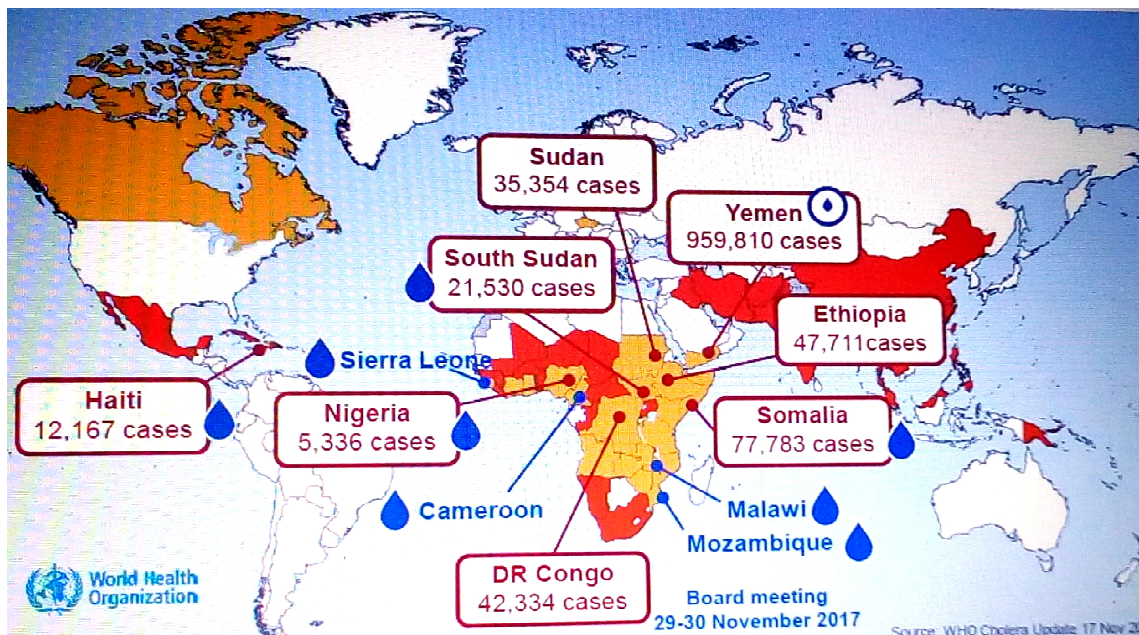


Fig. 1. Countries Affected BY Cholera In 2017

Source WHO Cholera update [15]

- Countries reporting cholera, 2010-2016
- Countries reporting cholera, 2017

Vibrio cholerae O139 Bengal, ushered in the eighth Cholera pandemic in 1992 [5]. It began in India and Bangladesh and then extended to South-East Asia, Africa and other parts of the world [39]. A surveillance report in Dhaka, Bangladesh, revealed a trend of increasing infection rate over past 3 decades though the fatality rate decreased [40,41].

4. THE TREND OF CHOLERA OUTBREAKS IN AFRICA (2010-2018)

In a cholera report by [24], they stated that between 2002 and 2011, African continent alone reported over two-thirds of the 2.2 million worldwide cases of cholera. WHO reports also stated that there has been a global resurgence of

cholera, causing approximately 120,000 deaths annually [42].

About 90% of the total global cases of cholera reported in 2010 were from Hispaniola (Haiti and the Dominican Republic), Lake Chad Basin (Nigeria, Cameroon, Niger, and Chad), and the region of the Great Lakes/ the eastern coast of Africa (East of the DRC, Uganda, Kenya, Burundi, the United Republic of Tanzania, Zambia, and Mozambique) [11].

Spreading along watercourses and worst still, during the rainy season, UNICEF identified three main cholera epidemic outbreaks in the Lake Chad basin, the West Congo basin and Lake Tanganyika, where about 90% of cases and fatalities were reported from Cameroon, Ghana, Nigeria, the Democratic Republic of the Congo (RDC) and Chad [43].

In Haiti, even though there had been no report of Cholera since 1910, a resurgence was reported [44,45]. This was marked by the cholera outbreak that occurred in October 2010, after the earthquake. As of July 7, 2011, >385,000 cases and 5,800 deaths were reported, from this outbreak [46].

Oger & Sudre [47] in their work in the Lake Chad basin on cholera, published the map from 1 January 2010 to 17 October 2010 involving Chad, Niger, Nigeria, and Cameroon. In this map, it showed that, the 1st of January to 4th of April, was termed the Inter-epidemic period, and only a few cases persisted in three local government areas of Nigeria. According to this report, the epidemic started in Cameroon (Mayo-Sava and Mayo-Tsanaga, Mayo-Danay areas), Adamawa (Nigeria) and Niger from the 5th of April to 4th of July and from the 5th of July to 22nd August there was a marked increase in cases but fresh areas were reported. The spread of the epidemic to other areas and the highest number of cases was only recorded from the 23rd of August to 17th October 2010 [24].

However, in the same 2010, a total of 110,115 cases were reported by [48] from sub-Saharan African countries, a 46% decline compared to 2009. They also stated that Cameroon, Chad, Niger and Nigeria, recorded about 54% (62,762) of cases and 77% (2,610) of deaths during this time. Niger reported 38% of this (44,456 cases), a case fatality rate (CFR) of 3.9%. This was beyond the 2000–2005 report of the African

mean overall CFR and the acceptable WHO rate of 2.4% and 1% respectively [34,48].

On the other hand, an O1 cholera epidemic that was reported in the Republic of Guinea, stated that, the first cases were notified in the prefecture of Forécariah in February [49]. They noted that, close to 25,000 cases and 392 deaths were recorded, the worst registered after about 15years in Guinea and Sierra Leone.

In Nigeria, [11] sited that Nigeria belongs to the group of three major current cholera foci in the world. In, 2010Borno State experienced a severe outbreak of cholera which later spread throughout the country [12]. According to WHO report, this epidemic, (the worst in Nigeria since 1991), left 7,654 people dead out of 59,478 cases [13]. This report has it that, in 2010 and 2011, Nigeria recorded 6,400 and 23,366 cases of cholera with 352 and 742 deaths respectively.

Ayeni [15] reported the death of 3 persons and 42 hospitalized cases in a cholera outbreak that occurred in Ede North and South LGAs of South-western Nigeria in November 2012. An outbreak also occurred in a sub-Urban area of Akwanga L.G.A of Nasarawa State, North Central in September 12th to 14th, 2013 [14].

An outbreak of cholera was confirmed in 14 states in Nigeria, in the first quarter of 2014 with a total of 9,006 cases and 106 deaths recorded; among which 855 cases, 17 lab confirmed and 20 deaths (Case Fatality Rate 3.3%) were reported from 28 Local Government Areas (LGAs), in 9 States as of 31 January 2014 This was lower than what was recorded during the same period in 2012 and 2013 [16]. However, an upward trend of cases was observed three weeks later, when 288 cases and 10 deaths were reported in four LGAs from Ebonyi, Federal Capital Territory (FCT), Kano and above all, Bauchi State where about 237 (79.5%) of these reported cases of January 2014, were reported from although no death was recorded. This is almost a repeat of the trend of event observed in Bauchi State, at about the same period in 2013 [13].

Reports of the presence of Ogawa serotypes and Classical biotype but predominantly the El Tor biotype in the 2010 outbreak in North-Eastern Nigeria was just a confirmation of that by [13] who stated that, *V. cholerae* O1 strains circulating in Nigeria include both Inaba and Ogawa. They said that a possibility exists that

the Inaba strain could have originated from the endemic strain while the outbreak was caused by the Ogawa strain. This report was further ascertained by the work of [12] that performed a comprehensive characterization of representative *V. cholerae* strains from sequential outbreaks in Nigeria and reported that recent cholera outbreaks in Nigeria are driven by atypical El Tor strains.

Kolawole et al. [13], published a reports which stated that, the *wbtT* gene, which determines the specificity of Ogawa, is involved in the serotype switching observed in the Nigerian epidemic *Cholera* O1 strains, and that the El Tor strains carrying the cholera toxin gene (*ctx*) of the Classical type were isolated among the Nigerian epidemic *V. cholera*, and designated El Tor variants.

Following the progressive report of cholera in the border states of Adamawa, Borno and Taraba Nigeria in 2009, Cameroon, (Garoua and Maroua areas) also recorded 144 cases and 51 deaths [17,18]. In this report, by the end of August 2010, the Extreme North and North regions of Cameroon followed the cue while cases were also confirmed in Douala in the same year. In the report by [50], total of about 10,741 people were affected in the Cameroon and 650 died; Approximately 9,406 cases and 600 deaths in Far North region, 511 cases and 24 deaths in North region and 457 cases and 12 deaths in the Littoral region. South West region registered 336 cases and 13 deaths, the Centre region registered 30 cases and 1 death, and the Adamawa region registered 1 case and no death.

The wave of epidemic continued in 2011, with 9 regions out of 10 affected in Cameroon. The regions of Adamawa, North West and South West, recorded the highest cases (22,762 cases) and 786 fatalities [18]. From the 7th week of 2011, 17 cases of cholera were suddenly registered in the capital Yaoundé, and other parts of the Centre region. The number of cases remained on the increasing side, reaching 687 cases and 43 deaths in Centre region by 29 March [50].

Following the Global Health Observatory Reports [51] 172 454 cases of cholera were recorded in 2015, and out of this number, 6 died. A 23% reduction in this number of cases was recorded in 2016 (132,121 cases), from which Africa reported 54% [52] and Kenya alone,

registered 11,033 cases by the 15th of January 2016 [53].

However, a sharp increase in the case fatality rates above this record was observed in 2016. A similar trend of events was observed in 2017, especially from different states of Nigeria where there was a steady increase in the number of cases as the weeks passed by. The New Telegraph Newspaper [54], in a report published on the 25th of June 2018, asserted that, in 2017, 4,221 cases of cholera were recorded in 20 of the Nigeria's 36 states. However, 107 of the cases died and only 60 out of this number of cases were successfully confirmed as being cholera. In Kwara state, the total number of registered cases rose to 1,178, plus 9 deaths by the 14th of June 2017, and the agent responsible for this was confirmed to be *V. cholera* O1 [55,56]. In Borno State, the population affected was lesser than that of Kwara State, although, the number of deaths was more (152 unconfirmed cases, and 11 deaths, by the 31st of August 2017 from Muna Garage) [57]. According to the WHO [55] reports, in Africa, cholera burden was such that Nigeria > than Ghana > Sudan.

In 2018, more than 50 deaths out of the 2,479 recorded cases were registered in 16 states. However, only 78 of the cases were actually confirmed as cholera. Furthermore, in a similar report from NCDC on the 16 states of Nigeria, said 16008 unconfirmed cholera cases and 186 deaths were recorded from January to the 8th of July 2018 [58]. The bulk of these cases (1564) were reported from Adamawa state; where the WHO news agency of Nigeria, in their summary of epidemiologic reports, stated that from the first of May to twenty- second of June 2018, 1,388 cases including 25 deaths were reported in Hong, Maiha, Mubi North and Mubi South [59,52].

From the News Agency of Nigeria [60], cholera report on Katsina state, Muhamadu Abubakar stated that, six local Governments were struck by the epidemic; and by July 2018, 11 people were reported dead in Funtua and Kusada local governments.

Similarly, in Niger state, about 60 cases and 15 deaths were reported in Bida lga by Sheriff Shitu [61] in 2018, in an outbreak.

Other African countries were not left out of the cholera epidemic picture in 2018. The alarm of

new cases came by the 23rd of March 2018, from S. Africa and Uganda. The trend of cholera reported cases from African countries since the beginning of the outbreak in 2016 was such that Yemen recorded more than one million cases (17,788 cases and 8 deaths in 2018 alone). This was followed by DR Congo with 2,688 cases and 85 deaths, then by Uganda with 1,696 cases and 36 deaths, Kenya with 1,277 cases and lastly Zambia, 1,241 cases and 19 deaths. However, in Haiti, the case was different because a decreasing trend was observed (281 cases and seven deaths) [62].

5. ROUTES OF CHOLERA SPREAD TO COASTAL AFRICA AND INHERENT CONTRIBUTORY FACTORS

Beginning along India's Ganges River as an epidemic outbreak in 1817, the initial cholera epidemic traveled by back and forth or barter routes, through waterways contaminated by sewage [6], spreading through British ships and battalion migration in 1826. The globalization of the world by exhilarating systems and increased elatedness of religious pilgrims diversified the rate, harshness and unpleasant effects usually observed in pandemics.

By 1830 *Vibrio cholerae* appeared in the Baltic, England, Ireland, Canada, USA and Mexico conveyed by Russian soldiers, English sailors, people en-route to Ireland and Canadian scientists. Then, in 1831 through Muslim pilgrims it entered Mecca and killed the Mecca and Jeddah governors alongside many others [63,64]. The Kansas Historical Society [65], has it that the transmission of cholera to troopers at Jefferson Barracks through contaminated incoming ships en route to Canada through St. Lawrence River,. Sailors also brought the cholera to Missouri and Joseph La Barge, a cholera survivor who dwelled in this city, was again reminded of the trauma of haven buried eight corpses of cholera victims in one grave just below Kansas City. The dread of a cholera epidemic threw the Missouri inhabitants in a pandemonium that led to an attempt to so destroy the ship [65]. This paper further stated that, cattle rearers traversing through the Pacific coast, further spread the superbug of a disease in these areas, right down to South and Central America. Mukhopadhyay et al. [7], reported that in 1905, at a quarantine station in El Tor Egypt, a German physician, E. Gotschlic first isolated *V. cholerae* O1 El Tor, from Indonesian pilgrims on their way to Mecca. From Indonesia, it spread in

1961 through Asia to Africa (Guinea) in 1970 [46, 2]. From September 1970, the chronological invasion of west African countries began, starting with Guinea [66], then Sierra Leone, Liberia, Cote d'ivoire, Mali, Togo, Dahomey, Upper Volta (Burki Na-faso) and finally Nigeria (Lagos) and Niger in December, 1970 [67,47,24,34,68].

Invariably the disease has become endemic in Africa and for more than 10 years, the coastal regions have been the hot spots; facilitated by marine trade and transport [33]. Furthermore, epidemics have been reported across the boundaries to other countries; for example, Liberia and Sierra Leone to Guinea (1994, 2003–2007 and 2012). From 2006–2007, it was from Luanda to Angola, Congo and from Pointe-Noire (Congo) to Brazzaville. In 1999, it was from Comoros to Mahajunga and the rest of Madagascar. In 1994 and 1999, the across the boundary epidemic was from Djibouti to Somalia. In 1994–1995, it was from Guinea-Bissau to Senegal. In 1993 and 1998, it was from Djibouti to Ethiopia. In 1991, it crossed from Cotonou to northern Benin. In 1980–1981 and 1997 it trespassed from Mozambique to KwaZulu-Natal and the rest of South Africa [34].

Political unrest, urbanization, poverty, under-development, natural disasters e.g earthquakes, floods tsunamis, Hurricanes etc and the availability of the faster global transport systems may have contributed to the dissemination of the infection [7]

Displaced people traveling from affected regions to their various homes especially after the 2010 Haitian outbreak also became faster vehicles of transmission and spread to other areas in Africa, like those long African coasts (such as the estuarine cities of Bissau in Guinea-Bissau, Calabar in Nigeria, Douala in Cameroon, Beira in Mozambique, or Dar es Salam in Tanzania). But no good and elaborate report concerning the routes of spread of cholera to these presumptive coastal African hotspots has been documented except for the Bay of Bengal [34].

6. CONCLUSION

Statistically, cholera burden and magnitude has been shown to be significantly associated with the nearness to riversides as well as areas that have a link with the marine environment. The

route of spread, alongside other factors like environmental and demographic factors, the non-pathogenic environmental strains (their invasiveness, virulence and pathogenicity characteristics), may help in the prediction of future outbreaks of the disease and for the preparation of better prevention and intervention strategies. The development of better surveillance and prompt intervention strategies in such cholera sickened areas as well as better waste disposal and management systems, portable water systems, adequate medical and healthcare facilities etc, will go a long way to curb this disease in such areas of globe.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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